

\$%^STN;Highlighton= ***;Highlightoff=***

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

10.072-15%

LOGINID:SSSPTA1647RBK

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

NEWS	1	Web Page URLs for STN Seminar Schedule - N. America
NEWS	2	"Ask CAS" for self-help around the clock
NEWS	3	SEP 01 New pricing for the Save Answers for SciFinder Wizard within STN Express with Discover!
NEWS	4	OCT 28 KOREAPAT now available on STN
NEWS	5	NOV 18 Current-awareness alerts, saved answer sets, and current search transcripts to be affected by CERAB, COMPUAB, ELCOM, and SOLIDSTATE reloads
NEWS	6	NOV 30 PHAR reloaded with additional data
NEWS	7	DEC 01 LISA now available on STN
NEWS	8	DEC 09 12 databases to be removed from STN on December 31, 2004
NEWS	9	DEC 15 MEDLINE update schedule for December 2004
NEWS	10	DEC 17 ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	11	DEC 17 COMPUAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	12	DEC 17 SOLIDSTATE reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	13	DEC 17 CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	14	DEC 17 THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS EXPRESS		OCTOBER 29 CURRENT WINDOWS VERSION IS V7.01A, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0jc(JP), AND CURRENT DISCOVER FILE IS DATED 11 AUGUST 2004
NEWS HOURS		STN Operating Hours Plus Help Desk Availability
NEWS INTER		General Internet Information
NEWS LOGIN		Welcome Banner and News Items
NEWS PHONE		Direct Dial and Telecommunication Network Access to STN
NEWS WWW		CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

FILE 'HOME' ENTERED AT 15:57:21 ON 18 DEC 2004

=> file medline biosis caplus scisearch
COST IN U.S. DOLLARS

COST IN DOLLARS		SINCE FILE ENTRY	TOTAL SESSION 0.21
FULL ESTIMATED COST		0.21	0.21

FILE 'MEDLINE' ENTERED AT 15:57:37 ON 18 DEC 2004

FILE 'BIOSIS' ENTERED AT 15:57:37 ON 18 DEC 2004
Copyright (c) 2004 The Thomson Corporation.

FILE 'CAPLUS' ENTERED AT 15:57:37 ON 18 DEC 2004
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (c1q or adipoq or apm1 or (acrp(w)30) or cerebellin or multimerin)
L1 10959 (C1Q OR ADIPOQ OR APM1 OR (ACRP(W) 30) OR CEREBELLIN OR MULTIMER
IN)
=> s lipolysis(w)stimulated(w)receptor
L2 83 LIPOLYSIS(W) STIMULATED(W) RECEPTOR
=> s obesity(s)(type(w)(2 or II)(w)diabetes)
L3 6417 OBESITY(S)(TYPE(W)(2 OR II)(W) DIABETES)
=> s l3(p)(treatment or therapy or therapeutic or treating or administer?)
L4 1992 L3(P)(TREATMENT OR THERAPY OR THERAPEUTIC OR TREATING OR ADMINI
STER?)
=> s l4 and l1
L5 6 L4 AND L1
=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 2 DUP REM L5 (4 DUPLICATES REMOVED)

=> d ibib abs 102
2 ANSWERS ARE AVAILABLE. SPECIFIED ANSWER NUMBER EXCEEDS ANSWER SET SIZE
The answer numbers requested are not in the answer set.
ENTER ANSWER NUMBER OR RANGE (1):1-2

L6 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003548073 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14626649
TITLE: [Adiponectin: a new adipocytokine].
L'adiponectine: une nouvelle adipocytokine.
AUTHOR: Lebas E; Paquot N; Scheen A J
CORPORATE SOURCE: Service de Diabetologie, Nutrition et Maladies
metaboliques, Departement de Medecine, CHU Sart Tilman,
Liege.
SOURCE: Revue medicale de Liege, (2003 Sep) 58 (9) 554-8. Ref: 21
Journal code: 0404317. ISSN: 0370-629X.
PUB. COUNTRY: Belgium
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20031121
Last Updated on STN: 20040416
Entered Medline: 20040415

AB Adipose tissue is not simply a store of excess energy, but also secretes a variety of proteins into circulating blood that influence systemic metabolism. These include tumor necrosis factor (TNF-alpha), plasminogen activator inhibitor type 1 (PAI-1), leptin, resistine and adiponectin. These are collectively known as adipocytokines. Adiponectin (also referred to as ***AdipoQ***, ***Acrp***, ***30***, ***apM1*** or GBP28) is a novel adipose-specific protein. A recent genome study mapped a susceptibility locus for type 2 diabetes and the metabolic syndrome on chromosome 3q27, where the adiponectin gene is located. Adiponectin is a peculiar adipocytokine because in contrast to the markedly increased levels of many others, as leptin or TNF-alpha, its level is reduced in ***obesity*** and ***type***, ***2***, ***diabetes***. The administration of thiazolidinediones, which are synthetic PPARs-gamma ligands, significantly increases the plasma adiponectin concentrations, an effect that could improve insulin sensitivity. Thus, the administration of adiponectin may provide a novel ***treatment*** modality for insulin resistance and type 2 diabetes.

L6 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2003099617 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12611609
TITLE: The role of the novel adipocyte-derived hormone adiponectin in human disease.
AUTHOR: Diez Juan J; Iglesias Pedro
CORPORATE SOURCE: Department of Endocrinology, Hospital Ramon y Cajal,
Madrid, Spain.. mibarsd@infomed.es

SOURCE: European journal of endocrinology / European Federation of Endocrine Societies, (2003 Mar) 148 (3) 293-300. Ref: 72
Journal code: 9423848. ISSN: 0804-4643.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030304
Last Updated on STN: 20030419
Entered Medline: 20030418

AB Adiponectin, also called GBP-28, ***apM1***, ***AdipoQ*** and Acrp30, is a novel adipose tissue-specific protein that has structural homology to collagen VIII and X and complement factor ***C1q***, and that circulates in human plasma at high levels. It is one of the physiologically active polypeptides secreted by adipose tissue, whose multiple functions have started to be understood in the last few years. A reduction in adiponectin expression is associated with insulin resistance in some animal models. Administration of adiponectin has been accompanied by a reduction in plasma glucose and an increase in insulin sensitivity. In addition, thiazolidinediones, drugs that enhance insulin sensitivity through stimulation of the peroxisome proliferator-activated receptor-gamma, increase plasma adiponectin and mRNA levels in mice. On the other hand, this adipocyte protein seems to play a protective role in experimental models of vascular injury. In humans, adiponectin levels are inversely related to the degree of adiposity and positively associated with insulin sensitivity both in healthy subjects and in diabetic patients. Plasma adiponectin levels have been reported to be decreased in some insulin-resistant states, such as ***obesity*** and ***type***
2 ***diabetes*** mellitus, and also in patients with coronary artery disease. On the contrary, chronic renal failure, type 1 diabetes and anorexia nervosa are associated with increased plasma adiponectin levels. Concentrations of plasma adiponectin have been shown to correlate negatively with glucose, insulin, triglyceride levels and body mass index, and positively with high-density lipoprotein-cholesterol levels and insulin-stimulated glucose disposal. Weight loss and ***therapy*** with thiazolidinediones increased endogenous adiponectin production in humans. Adiponectin increases insulin sensitivity by increasing tissue fat oxidation, resulting in reduced circulating fatty acid levels and reduced intracellular triglyceride contents in liver and muscle. This protein also suppresses the expression of adhesion molecules in vascular endothelial cells and cytokine production from macrophages, thus inhibiting the inflammatory processes that occur during the early phases of atherosclerosis. In view of these data, it is possible that hypoadiponectinemia may play a role in the development of atherosclerotic vascular disease. In summary, the ability of adiponectin to increase insulin sensitivity in conjunction with its anti-inflammatory and anti-atherogenic properties have made this novel adipocytokine a promising ***therapeutic*** tool for the future, with potential applications in states associated with low plasma adiponectin levels.

L16 ANSWER 1 OF 2 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2003:718325 SCISEARCH

THE GENUINE ARTICLE: 711HH

TITLE: Glucose-to-insulin ratio rather than sex hormone-binding globulin and ***adiponectin*** levels is the best predictor of insulin resistance in Nonobese women with polycystic ovary syndrome

AUTHOR: Ducluzeau P H; Cousin P; Malvoisin E; Bornet H; Vidal H; Laville M; Pugeat M (Reprint)

CORPORATE SOURCE: Hospices Civils Lyon, Hop Neurocardiol, Federat Endocrinol, Batiment HGPO, 59 Blvd Pinel, F-69349 Lyon 03, France (Reprint); Hospices Civils Lyon, Hop Eouard Herriot, Federat Biochim, F-69437 Lyon 08, France; Hospices Civils Lyon, Ctr Rech Nutr Humaine, F-69437 Lyon, France; Fac Med Laennec, INSERM, U449, F-69373 Lyon 08, France; Hop Antiquaille, Federat Endocrinol, F-69321 Lyon 05, France

COUNTRY OF AUTHOR:

France

SOURCE:

JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (AUG

2003) Vol. 88, No. 8, pp. 3626-3631.

Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE

500, BETHESDA, MD 20814-4110 USA.

ISSN: 0021-972X.

DOCUMENT TYPE:

Article; Journal

LANGUAGE:

English

REFERENCE COUNT:

44

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Polycystic ovary syndrome (PCOS), the main androgen disorder in women, has been suggested to be associated with a high risk of developing cardiovascular disease and ***type*** ***2*** ***diabetes***. In many PCOS patients, overweight or central ***obesity*** is generally associated with increases in fasting insulin levels, insulin resistance, and glucose intolerance, and has been identified as a target for new ***therapeutic*** strategy, including early change in lifestyle. Early biochemical marker(s) for identifying at-risk patients will be useful for prevention studies. The main goal of the present study was to search for such tool(s). We investigated 16 nonobese PCOS women by performing euglycemic hyperinsulinemic clamp and measuring insulin levels during fasting and oral glucose tolerance test, as well as the serum concentrations of SHBG, leptin, and ***adiponectin***, the newly identified adipose factors. Eight of the 16 patients had a steady-state glucose disposal rate less than 8.5 mg/kg.min, the lowest normal value for nonobese control women. These insulin-resistant patients had significant higher body mass index (BMI) and waist-to-hip ratio (WHR), and lower high-density ***lipoprotein*** cholesterol and SHBG levels. As expected, glucose disposal correlated negatively with BMI ($P=0.01$), WHR ($P=0.01$), and fasting insulin level ($P=0.003$). On stepwise regression analysis, however, the glucose-to-insulin ratio (GIR) emerged as the strongest independent parameter to appraise insulin resistance ($R^2=0.61$). SHBG level correlated positively with GIR ($P<0.001$) and negatively with BMI ($P=0.003$) but did not correlate with either insulin response during the glucose tolerance test or ***plasma*** leptin and/or ***adiponectin*** levels. In contrast, BMI was the only independent predictive parameter of SHBG ($P=0.003$, $R^2=0.73$). Interestingly, ***plasma*** ***adiponectin*** levels were positively associated with glucose disposal rate ($P=0.043$) and negatively with WHR ($P=0.024$), waist circumference being the best predictor of ***adiponectin*** level ($P<0.01$). Leptin level correlated only with BMI ($r=0.62$, $P=0.01$). This study confirmed that insulin resistance, despite the lack of ***obesity*** as such, is clearly present in many PCOS women, and demonstrated that GIR is the best predictor for insulin resistance. It was also shown that ***adiponectin*** level is a good indicator of abdominal fat mass and is associated to insulin resistance. Finally, low SHBG levels in PCOS are intimately associated with BMI, suggesting that some signal(s) from the adipose tissue, independent of ***adiponectin*** and leptin, may regulate liver production of SHBG.

L16 ANSWER 2 OF 2

MEDLINE on STN

DUPLICATE 1

ACCESSION NUMBER:

2003099617 MEDLINE

DOCUMENT NUMBER:

PubMed ID: 12611609

TITLE:

The role of the novel adipocyte-derived hormone ***adiponectin*** in human disease.

AUTHOR:

Diez Juan J; Iglesias Pedro

CORPORATE SOURCE:

Department of Endocrinology, Hospital Ramon y Cajal, Madrid, Spain.. mibarsd@infomed.es

SOURCE:

European journal of endocrinology / European Federation of Endocrine Societies, (2003 Mar) 148 (3) 293-300. Ref: 72 Journal code: 9423848. ISSN: 0804-4643.

PUB. COUNTRY:

England: United Kingdom

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200304

ENTRY DATE:

Entered STN: 20030304

Last Updated on STN: 20030419

Entered Medline: 20030418

AB

Adiponectin, also called GBP-28, apM1, AdipoQ and Acrp30, is a novel adipose tissue-specific protein that has structural homology to collagen VIII and X and complement factor C1q, and that circulates in human plasma at high levels. It is one of the physiologically active polypeptides secreted by adipose tissue, whose multiple functions have

started to be understood in the last few years. A reduction in ***adiponectin*** expression is associated with insulin resistance in some animal models. Administration of ***adiponectin*** has been accompanied by a reduction in plasma glucose and an increase in insulin sensitivity. In addition, thiazolidinediones, drugs that enhance insulin sensitivity through stimulation of the peroxisome proliferator-activated receptor-gamma, increase plasma ***adiponectin*** and mRNA levels in mice. On the other hand, this adipocyte protein seems to play a protective role in experimental models of vascular injury. In humans,

adiponectin levels are inversely related to the degree of adiposity and positively associated with insulin sensitivity both in healthy subjects and in diabetic patients. Plasma ***adiponectin*** levels have been reported to be decreased in some insulin-resistant states, such as ***obesity*** and ***type*** 2 ***

diabetes mellitus, and also in patients with coronary artery disease. On the contrary, chronic renal failure, type 1 diabetes and anorexia nervosa are associated with increased plasma ***adiponectin*** levels. Concentrations of ***plasma*** ***adiponectin*** have been shown to correlate negatively with glucose, insulin,

triglyceride levels and body mass index, and positively with high-density ***lipoprotein*** -cholesterol levels and insulin-stimulated glucose disposal. Weight loss and ***therapy*** with thiazolidinediones increased endogenous ***adiponectin*** production in humans. ***Adiponectin*** increases insulin sensitivity by increasing tissue fat oxidation, resulting in reduced circulating fatty acid levels and reduced intracellular triglyceride contents in liver and muscle. This protein also suppresses the expression of adhesion molecules in vascular endothelial cells and cytokine production from macrophages, thus inhibiting the inflammatory processes that occur during the early phases of atherosclerosis. In view of these data, it is possible that hypoadiponectinemia may play a role in the development of atherosclerotic vascular disease. In summary, the ability of ***adiponectin*** to increase insulin sensitivity in conjunction with its anti-inflammatory and anti-atherogenic properties have made this novel adipocytokine a promising

therapeutic tool for the future, with potential applications in states associated with low plasma ***adiponectin*** levels.

L23 ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2004:356948 BIOSIS
DOCUMENT NUMBER: PREV200400363462
TITLE: T-cadherin is a receptor for hexameric and high-molecular-weight forms of Acrp30/adiponectin.
AUTHOR(S): Hug, Christopher; Wang, Jin; Ahmad, Naina Shehzeen; Bogan, Jonathan S.; Tsao, Tsu-Shuen; Lodish, Harvey F. [Reprint Author]
CORPORATE SOURCE: Whitehead Inst Biomed Res, 9 Cambridge Ctr, Cambridge, MA, 02142, USA
lodish@wi.mit.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (July 13 2004) Vol. 101, No. 28, pp. 10308-10313. print.
ISSN: 0027-8424 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Sep 2004
Last Updated on STN: 5 Sep 2004
AB Acrp30/adiponectin is reduced in the serum of obese and diabetic individuals, and the genetic locus of adiponectin is linked to the metabolic syndrome. Recombinant ***adiponectin***, ***administered*** to diet-induced obese mice, induced weight loss and improved insulin sensitivity. In muscle and liver, adiponectin stimulates AMP-activated protein kinase activation and fatty acid oxidation. To expression-clone molecules capable of binding adiponectin, we transduced a C2C12 myoblast cDNA retroviral expression library into Ba/F3 cells and panned infected cells on recombinant adiponectin linked to magnetic beads. We identified T-cadherin as a receptor for the hexameric and high-molecular-weight species of adiponectin but not for the trimeric or globular species. Only eukaryotically expressed adiponectin bound to T-cadherin, implying that posttranslational modifications of adiponectin are critical for binding. An adiponectin mutant lacking a conserved N-terminal cysteine residue required for formation of hexamer and high-molecular-weight species did not bind T-cadherin in coimmunoprecipitation studies. Although lacking known cellular functions,

T-cadherin is expressed in endothelial and smooth muscle cells, where it is positioned to interact with adiponectin. Because T-cadherin is a glycosylphosphatidylinositol-anchored extracellular protein, it may act as a coreceptor for an as-yet-unidentified signaling receptor through which adiponectin transmits metabolic signals.

L23 ANSWER 2 OF 5 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2004:1019767 SCISEARCH

THE GENUINE ARTICLE: 871DL

TITLE: Modulation of adipoinsular axis in prediabetic Zucker diabetic fatty rats by diazoxide

AUTHOR: Alemezadeh R (Reprint); Tushaus K M

CORPORATE SOURCE: Med Coll Wisconsin, Dept Pediat, Sect Endocrinol & Metab, 8701 Watertown Plank Rd, Milwaukee, WI 53226 USA (Reprint); Med Coll Wisconsin, Dept Pediat, Sect Endocrinol & Metab, Milwaukee, WI 53226 USA

COUNTRY OF AUTHOR: USA

SOURCE: ENDOCRINOLOGY, (DEC 2004) Vol. 145, No. 12, pp. 5476-5484. Publisher: ENDOCRINE SOC, 8401 CONNECTICUT AVE, SUITE 900, CHEVY CHASE, MD 20815-5817 USA.

ISSN: 0013-7227.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 59

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Dysregulation of the adipoinsular axis in male obese Zucker diabetic fatty (ZDF; fa/fa) rats, a model of type 2 ***diabetes***, results in chronic hyperinsulinemia and increased de novo lipogenesis in islets, leading to beta-cell failure and ***diabetes***. Diazoxide (DZ; 150 mg/kg.d), an inhibitor of insulin secretion, was ***administered*** to prediabetic ZDF animals for 8 wk as a strategy for prevention of ***diabetes***. DZ reduced food intake ($P<0.02$) and rate of weight gain only in ZDF rats ($P<0.01$). Plasma insulin response to glucose load was attenuated in DZ-Zucker lean rats (ZL; $P<0.01$), whereas DZ-ZDF had higher insulin response to glucose than controls ($P<0.001$). DZ improved hemoglobin A(1c) ($P<0.001$) and glucose tolerance in ZDF ($P<0.001$), but deteriorated hemoglobin A(1c) in ZL rats ($P<0.02$) despite normal tolerance in the fasted state. DZ lowered plasma leptin ($P<0.001$), free fatty acid, and triglyceride ($P<0.001$) levels, but increased ***adiponectin*** levels ($P<0.02$) only in ZDF rats. DZ enhanced beta(3)-adrenoreceptor mRNA ($P<0.005$) and adenylate cyclase activity ($P<0.01$) in adipose tissue from ZDF rats only, whereas it enhanced islet beta(3)-adrenergic receptor mRNA ($P<0.005$) but paradoxically decreased islet adenylate cyclase activity ($P<0.005$) in these animals. Islet fatty acid synthase mRNA ($P<0.03$), acyl coenzyme A carboxylase mRNA ($P<0.01$), uncoupling protein-2 mRNA ($P<0.01$), and triglyceride content ($P<0.005$) were only decreased in DZ-ZDF rats, whereas islet insulin mRNA and insulin content were increased in DZ-ZDF ($P<0.01$) and DZ-ZL rats ($P<0.03$). DZ-induced beta-cell rest improved the lipid profile, enhanced the metabolic efficiency of insulin, and prevented beta-cell dysfunction and ***diabetes*** in ***diabetes***-prone animals. This ***therapeutic*** strategy may be beneficial in preventing beta-cell failure and progression to ***diabetes*** in humans.

L23 ANSWER 3 OF 5 MEDLINE on STN

ACCESSION NUMBER: 2004267156 IN-PROCESS

DOCUMENT NUMBER: PubMed ID: 15168385

TITLE: Adiponectin in peritoneal dialysis patients: a comparison with hemodialysis patients and subjects with normal renal function.

AUTHOR: Huang Jenq-Wen; Yen Chung-Jen; Chiang Hong-Wei; Hung Kuan-Yu; Tsai Tun-Jun; Wu Kwan-Dun

CORPORATE SOURCE: Department of Internal Medicine, National Taiwan University Hospital and College of Medicine, Taipei, Taiwan.

SOURCE: American journal of kidney diseases : official journal of the National Kidney Foundation, (2004 Jun) 43 (6) 1047-55. Journal code: 8110075. ISSN: 1523-6838.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20040529

Last Updated on STN: 20040604

AB BACKGROUND: Adiponectin, a cytokine with anti-inflammatory properties that is secreted from adipose tissue, is associated with insulin resistance.

Adiponectin has been shown to be a predictor of cardiovascular events in both the general population and patients undergoing hemodialysis (HD); however, its role in peritoneal dialysis (PD) analogues remains unclear. METHODS: Serum adiponectin levels, measured by means of enzyme-linked immunosorbent assay in subjects with normal renal function and patients undergoing HD or PD (28 subjects in each group), were analyzed to establish the relationship between adiponectin and lipid levels, as well as insulin resistance. In the second study, 104 PD patients were recruited to analyze the relationships between serum adiponectin level and residual renal and peritoneal function and C-reactive protein (CRP) level. Independent factors for serum adiponectin level were determined from multiple linear regression. RESULTS: No significant difference was shown comparing serum adiponectin levels of PD and HD patients; however, both were significantly greater than those of control subjects ($P < 0.01$). Negative associations were shown between adiponectin and triglyceride (TG; $P < 0.01$) and insulin levels ($P < 0.05$) and homeostatic model assessment of insulin resistance ($P < 0.01$) for the former 2 groups; however, a positive association was shown for high-density lipoprotein (HDL) level ($P < 0.05$). Neither HD nor PD removed adiponectin significantly, with levels for the PD group negatively associated with residual renal function ($P < 0.01$) and CRP level ($P < 0.001$). PD patients ***administered*** glucose-lowering agents had lower ***adiponectin*** levels; however, lipid-lowering agents and renin-angiotensin blockades did not appear to affect them. Independent determinants for serum adiponectin level in PD patients were TG, HDL, and CRP levels and body mass index, after adjustment for age, sex, PD therapy duration, and ***diabetes***. Adiponectin levels were not associated with left ventricular mass or ejection fraction. As for HD patients, PD patients had high adiponectin levels; adiponectin was not removed effectively using either of the studied dialysis modalities. In addition to a significant relationship with the components of insulin resistance, adiponectin level was associated with CRP level in these patients. CONCLUSION: These results indicate that adiponectin level in PD patients may be a good indicator of cardiovascular disease risk.

L23 ANSWER 4 OF 5 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2003:1049140 SCISEARCH

THE GENUINE ARTICLE: 746AM

TITLE: Adiponectin in a native Canadian population experiencing rapid epidemiological transition

AUTHOR: Hanley A G (Reprint); Connelly P W; Harris S B; Zinman B
CORPORATE SOURCE: Mt Sinai Hosp, Leadership Sinai Ctr Diabet, 60 Murray St, Room 5210, Toronto, ON M5G 1X5, Canada (Reprint); Mt Sinai Hosp, Leadership Sinai Ctr Diabet, Toronto, ON M5G 1X5, Canada; Univ Toronto, Dept Med, Toronto, ON, Canada; Univ Toronto, Dept Publ Hlth, Toronto, ON, Canada; Univ Toronto, Dept Lab Med & Pathobiol, Toronto, ON, Canada; St Michaels Hosp, J Alick Little Lipid Res Lab, Toronto, ON, Canada; Univ Western Ontario, Ctr Studies Family Med, London, ON, Canada

COUNTRY OF AUTHOR: Canada

SOURCE: DIABETES CARE, (DEC 2003) Vol. 26, No. 12, pp. 3219-3225.
Publisher: AMER DIABETES ASSOC, 1701 N BEAUREGARD ST, ALEXANDRIA, VA 22311-1717 USA.

ISSN: 0149-5992.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 44

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB OBJECTIVE - Adiponectin is emerging as an important protein in the etiology of obesity and related metabolic disorders. The objectives of this study were to determine cross-sectional and prospective associations of adiponectin concentration with adiposity, type 2 ***diabetes***, and cardiovascular disease (CVD) risk factors in a population-based study of Native Canadians, a group experiencing dramatic increases in ***diabetes*** and CVD.

RESEARCH DESIGN AND METHODS - During the 1993-1995 baseline survey, samples for glucose, insulin, ***adiponectin***, and lipids were collected after an overnight fast. Waist circumference and percent body fat were measured, and a 75-g oral glucose tolerance test was

administered : n = 505 with normal glucose tolerance (NGT), 74 with impaired glucose tolerance (IGT), and 149 with ***diabetes***. In 1998, 95 high-risk subjects, defined as those who, at baseline, had either IGT or NGT with an elevated 2-h glucose concentration (greater than or equal to 7.0 mmol/l), participated, in a follow-up examination using the

protocol used at baseline.

RESULTS - After adjustment for covariates including percent body fat and homeostasis model assessment of insulin resistance (HOMA-IR), adiponectin concentrations were significantly lower among men versus women (10.8 vs. 15.0 $\mu\text{g}/\text{ml}$, $P < 0.0001$) and among diabetic versus NGT subjects (11.1 vs. 13.1 $\mu\text{g}/\text{ml}$, $P < 0.05$). Adiponectin was inversely correlated with percent body fat, waist circumference, HOMA-IR, and triglyceride and positively correlated with HDL ($r = 0.30 - 0.44$, all $P < 0.0001$). In multivariate linear regression analysis in nondiabetic adiponectin variation among subjects, HDL and percent body fat were significantly related to both men and women ($R^2 = 28-29\%$). Factor analysis returned three underlying factors among these variables, with adiponectin loading on the second factor along with insulin, waist circumference, triglyceride, and HDL. In the follow-up study, higher adiponectin at baseline was significantly associated with increases in HDL ($r = 0.24$, $P = 0.03$) and decreases in HOMA-IR ($r = -0.29$, $P = 0.009$) after adjustment for covariates, including age, adiposity, and ***diabetes*** status at baseline and follow-up.

CONCLUSIONS - These population-based findings support the hypothesis that low circulating levels of adiponectin are an important determinant of risk of CVD.

L23 ANSWER 5 OF 5 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2003:983576 SCISEARCH

THE GENUINE ARTICLE: 738PE

TITLE: Adiponectin: A regulator of energy homeostasis

AUTHOR: Wolf G (Reprint)

CORPORATE SOURCE: Univ Calif Berkeley, Dept Nutrit Sci & Toxicol, Berkeley, CA 94720 USA (Reprint)

COUNTRY OF AUTHOR: USA

SOURCE: NUTRITION REVIEWS, (AUG 2003) Vol. 61, No. 8, pp. 290-292. Publisher: INT LIFE SCIENCES INST NORTH AMERICA, ONE THOMAS CIRCLE, N W, 9TH FLOOR, WASHINGTON, DC 20005 USA. ISSN: 0029-6643.

DOCUMENT TYPE: General Review; Journal

LANGUAGE: English

REFERENCE COUNT: 19

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB ***Adiponectin***, a protein produced exclusively in adipose tissue, occurs in serum in relatively high concentration. Its concentration is decreased in obese and in type 2 diabetic humans. When ***administered*** to mice, it enhances insulin sensitivity and glucose tolerance, and appears to increase free fatty acid oxidation in muscle. ***Adiponectin*** is likely to be involved in the regulation of energy homeostasis.

L28 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2004:1063464 CAPLUS

TITLE: Relation between plasma resistin and adiponectin or

insulin resistance in type 2 diabetes mellitus

AUTHOR(S): Li, Chunrui; Zhang, Muxun; Liu, Wenli; Lu, Huiling;

Wang, Hongwei

CORPORATE SOURCE: Tongji Medical College, Huazhong University of Science and Technology, Wuhan, 430030, Peop. Rep. China

SOURCE: Wuhan Daxue Xuebao, Yixueban (2004), 25(5), 517-520

CODEN: WDXYAA; ISSN: 1671-8852

PUBLISHER: Wuhan Daxue Qikanshe

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB Eighty-eight subjects who were either completely healthy (28 cases) or had suffered from a ***type*** ***2*** ***diabetes*** (30 cases) or ***type*** ***2*** ***diabetes*** with ***obesity*** (30 cases) were recruited from Wuhan. All measurements (independent variables) were taken at the same time after clinic data were collected and followed by collection of fasting blood samples for anal. The ***plasma*** resistin levels pos. correlated with fasting ***plasma*** glucose (FPG), PG1h, PG2h in oral glucose tolerance test (OGTT), ***triglycerides*** and insulin resistance index (Homa-IR), neg. correlated with fasting ***plasma*** ***adiponectin***. Using step regression anal., BMI were significantly pos. related while ***adiponectin*** was closely neg. related to the plasma resistin levels after adjustment for all other variables in the model. The plasma

adiponectin levels had a neg. correlation with age, BMI, FPG, PG1h, PG2h, Homa-IR, leptin and resistin. Resistin and ***adiponectin*** correlates with ***obesity*** and ***type*** ***2*** ***diabetes*** mellitus.

L28 ANSWER 2 OF 9 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004425772 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15331202
TITLE: Oolong tea increases ***plasma*** ***adiponectin*** levels and low-density ***lipoprotein*** particle size in patients with coronary artery disease.
AUTHOR: Shimada Kenei; Kawarabayashi Takahiko; Tanaka Atsushi; Fukuda Daiju; Nakamura Yasuhiro; Yoshiyama Minoru; Takeuchi Kazuhide; Sawaki Tetsuya; Hosoda Kazuaki; Yoshikawa Junichi
CORPORATE SOURCE: Department of Internal Medicine and Cardiology, Graduate School of Medicine, Osaka City University Medical School, 1-4-3 Asahimachi, Abeno-ku, Osaka 545-8585, Japan.. shimadak@msic.med.osaka-cu.ac.jp
SOURCE: Diabetes research and clinical practice, (2004 Sep) 65 (3) 227-34.
PUB. COUNTRY: Journal code: 8508335. ISSN: 0168-8227. Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040828
Last Updated on STN: 20041022

AB BACKGROUND: Oolong tea has been studied for its effect on cardiovascular disease and obesity. Plasma ***adiponectin*** levels are reduced in ***obesity***, in patients with ***type*** ***2*** ***diabetes*** mellitus and in coronary artery disease (CAD).
OBJECTIVE: To investigate prospectively, whether intake of Oolong tea influences ***plasma*** ***adiponectin*** levels, low-density ***lipoprotein*** (LDL) particle size, total cholesterol, high-density ***lipoprotein*** (HDL) cholesterol, LDL cholesterol, serum ***triglyceride*** and ***plasma*** glucose levels in patients with CAD.
METHODS: Twenty two patients in our study consumed Oolong tea (1000 ml) or water for 1 month in our randomized cross-over study design.
RESULTS: There was a significant difference in plasma ***adiponectin*** levels before and after 1 month intake of Oolong tea (6.26 +/- 3.26 microg/ml versus 6.88 +/- 3.28 microg/ml, P < 0.05), and in plasma level LDL particle size (25.02 +/- 0.67 nm versus 25.31 +/- 0.60 nm, P < 0.01). The water-consuming control group showed no changes (6.28 +/- 3.28 microg/ml versus 6.23 +/- 3.21 microg/ml) in ***adiponectin*** levels or LDL particle sizes (25.03 +/- 0.70 nm versus 25.02 +/- 0.72 nm). We also observed a significant difference in hemoglobin A1c levels (7.23 +/- 4.45% versus 6.99 +/- 4.30%, P < 0.05) before and after intake of oolong tea.
CONCLUSION: Oolong tea may have beneficial effects on the progression of atherosclerosis in patients with CAD.

L28 ANSWER 3 OF 9 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004214490 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15052336
TITLE: Diabetes, lipids, and adipocyte secretagogues.
AUTHOR: Faraj May; Lu Hui Ling; Cianflone Katherine
CORPORATE SOURCE: Mike Rosenbloom Laboratory for Cardiovascular Research, McGill University Health Centre, Royal Victoria Hospital, Montreal, QC, Canada.
SOURCE: Biochemistry and cell biology = Biochimie et biologie cellulaire, (2004 Feb) 82 (1) 170-90. Ref: 283
Journal code: 8606068. ISSN: 0829-8211.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200411
ENTRY DATE: Entered STN: 20040429
Last Updated on STN: 20041103
Entered Medline: 20041102

AB That ***obesity*** is associated with insulin resistance and ***type*** ***II*** ***diabetes*** mellitus is well accepted. Overloading of white adipose tissue beyond its storage capacity leads to lipid disorders in non-adipose tissues, namely skeletal and cardiac muscles, pancreas, and liver, effects that are often mediated through

increased non-esterified fatty acid fluxes. This in turn leads to a tissue-specific disordered insulin response and increased lipid deposition and lipotoxicity, coupled to abnormal ***plasma*** metabolic and (or) ***lipoprotein*** profiles. Thus, the importance of functional adipocytes is crucial, as highlighted by the disorders seen in both "too much" (obesity) and "too little" (lipodystrophy) white adipose tissue. However, beyond its capacity for fat storage, white adipose tissue is now well recognised as an endocrine tissue producing multiple hormones whose plasma levels are altered in obese, insulin-resistant, and diabetic subjects. The consequence of these hormonal alterations with respect to both glucose and lipid metabolism in insulin target tissues is just beginning to be understood. The present review will focus on a number of these hormones: acylation-stimulating protein, leptin, ***adiponectin***, tumour necrosis factor alpha, interleukin-6, and resistin, defining their changes induced in obesity and diabetes mellitus and highlighting their functional properties that may protect or worsen lipid metabolism.

L28 ANSWER 4 OF 9 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2003:718325 SCISEARCH

THE GENUINE ARTICLE: 711HH

TITLE: Glucose-to-insulin ratio rather than sex hormone-binding globulin and adiponectin levels is the best predictor of insulin resistance in Nonobese women with polycystic ovary syndrome

AUTHOR: Ducluzeau P H; Cousin P; Malvoisin E; Bornet H; Vidal H; Laville M; Pugeat M (Reprint)

CORPORATE SOURCE: Hospices Civils Lyon, Hop Neurocardiol, Federat Endocrinol, Batiment HGPO, 59 Blvd Pinel, F-69349 Lyon 03, France (Reprint); Hospices Civils Lyon, Hop Eouard Herriot, Federat Biochim, F-69437 Lyon 08, France; Hospices Civils Lyon, Ctr Rech Nutr Humaine, F-69437 Lyon, France; Fac Med Laennec, INSERM, U449, F-69373 Lyon 08, France; Hop Antiquaille, Federat Endocrinol, F-69321 Lyon 05, France

COUNTRY OF AUTHOR: France

SOURCE: JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (AUG 2003) Vol. 88, No. 8, pp. 3626-3631.

Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4110 USA.

ISSN: 0021-972X.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 44

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Polycystic ovary syndrome (PCOS), the main androgen disorder in women, has been suggested to be associated with a high risk of developing cardiovascular disease and ***type*** ***2*** ***diabetes***. In many PCOS patients, overweight or central ***obesity*** is generally associated with increases in fasting insulin levels, insulin resistance, and glucose intolerance, and has been identified as a target for new therapeutic strategy, including early change in lifestyle. Early biochemical marker(s) for identifying at-risk patients will be useful for prevention studies. The main goal of the present study was to search for such tool(s). We investigated 16 nonobese PCOS women by performing euglycemic hyperinsulinemic clamp and measuring insulin levels during fasting and oral glucose tolerance test, as well as the serum concentrations of SHBG, leptin, and ***adiponectin***, the newly identified adipose factors. Eight of the 16 patients had a steady-state glucose disposal rate less than 8.5 mg/kg.min, the lowest normal value for nonobese control women. These insulin-resistant patients had significant higher body mass index (BMI) and waist-to-hip ratio (WHR), and lower high-density ***lipoprotein*** cholesterol and SHBG levels. As expected, glucose disposal correlated negatively with BMI ($P=0.01$), WHR ($P=0.01$), and fasting insulin level ($P=0.003$). On stepwise regression analysis, however, the glucose-to-insulin ratio (GIR) emerged as the strongest independent parameter to appraise insulin resistance ($R^2=0.61$). SHBG level correlated positively with GIR ($P<0.001$) and negatively with BMI ($P=0.003$) but did not correlate with either insulin response during the glucose tolerance test or ***plasma*** leptin and/or ***adiponectin*** levels. In contrast, BMI was the only independent predictive parameter of SHBG ($P=0.003$, $R^2=0.73$). Interestingly, ***plasma*** ***adiponectin*** levels were positively associated with glucose disposal rate ($P=0.043$) and negatively with WHR ($P=0.024$), waist circumference being the best predictor of ***adiponectin*** level ($P<0.01$). Leptin level correlated only with BMI ($r=0.62$, $P=0.01$).

This study confirmed that insulin resistance, despite the lack of ***obesity*** as such, is clearly present in many PCOS women, and demonstrated that GIR is the best predictor for insulin resistance. It was also shown that ***adiponectin*** level is a good indicator of abdominal fat mass and is associated to insulin resistance. Finally, low SHBG levels in PCOS are intimately associated with BMI, suggesting that some signal(s) from the adipose tissue, independent of ***adiponectin*** and Leptin, may regulate liver production of SHBG.

L28 ANSWER 5 OF 9 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2003330856 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12861229
TITLE: Adiponectin mRNA levels in the abdominal adipose depots of nondiabetic women.
AUTHOR: Yang W-S; Chen M-H; Lee W-J; Lee K-C; Chao C-L; Huang K-C; Chen C-L; Tai T-Y; Chuang L-M
CORPORATE SOURCE: Division of Endocrinology and Metabolism, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan.
SOURCE: International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity, (2003 Aug) 27 (8) 896-900.
Journal code: 9313169. ISSN: 0307-0565.

PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 20030716
Last Updated on STN: 20030826
Entered Medline: 20030825

AB BACKGROUND: The human ***adiponectin*** gene has been implicated in the pathophysiology of ***obesity***, ***type*** ***II*** ***diabetes*** mellitus, dyslipidemia and atherosclerosis. Investigation of the physiological functions of the ***adiponectin*** gene in humans was mainly conducted at the levels of plasma proteins or DNA polymorphisms. The depot-specific ***adiponectin*** mRNA levels also could be relevant to these physiological functions. OBJECTIVES: The relation between the adipose depot-specific ***adiponectin*** mRNA expression levels and various metabolic factors, including BMI, fasting ***plasma*** glucose, insulin, ***triglycerides*** (TGs) and HDL-cholesterol and insulin resistance index by HOMA, was investigated among 66 nondiabetic women using quantitative real-time PCR. RESULTS: The subcutaneous relative ***adiponectin*** mRNA levels (SRAmR) correlated significantly with the omental relative ***adiponectin*** mRNA levels (ORAmR) ($\gamma=0.468$, $P=0.0001$). The SRAmR correlated inversely with the fasting plasma glucose with a borderline significance ($\gamma=-0.35$, $P=0.058$). On the other hand, the ORAmR correlated negatively with serum TG levels with the adjustment for age ($\gamma=-0.33$, $P=0.007$) or age plus BMI ($\gamma=-0.27$, $P=0.027$). CONCLUSION: These results indicate that the ***adiponectin*** mRNA levels in different adipose depots were at least related to certain phenotypes of metabolic syndrome. The expression levels of ***adiponectin*** in the omental adipose depots are related to TG metabolism.

L28 ANSWER 6 OF 9 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2003099617 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12611609
TITLE: The role of the novel adipocyte-derived hormone adiponectin in human disease.
AUTHOR: Diez Juan J; Iglesias Pedro
CORPORATE SOURCE: Department of Endocrinology, Hospital Ramon y Cajal, Madrid, Spain.. mibarsd@infomed.es
SOURCE: European journal of endocrinology / European Federation of Endocrine Societies, (2003 Mar) 148 (3) 293-300. Ref: 72
Journal code: 9423848. ISSN: 0804-4643.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030304
Last Updated on STN: 20030419
Entered Medline: 20030418

AB ***Adiponectin***, also called GBP-28, apM1, AdipoQ and Acrp30, is a novel adipose tissue-specific protein that has structural homology to collagen VIII and X and complement factor C1q, and that circulates in human plasma at high levels. It is one of the physiologically active polypeptides secreted by adipose tissue, whose multiple functions have started to be understood in the last few years. A reduction in

adiponectin expression is associated with insulin resistance in some animal models. Administration of ***adiponectin*** has been accompanied by a reduction in plasma glucose and an increase in insulin sensitivity. In addition, thiazolidinediones, drugs that enhance insulin sensitivity through stimulation of the peroxisome proliferator-activated receptor-gamma, increase plasma ***adiponectin*** and mRNA levels in mice. On the other hand, this adipocyte protein seems to play a protective role in experimental models of vascular injury. In humans,

adiponectin levels are inversely related to the degree of adiposity and positively associated with insulin sensitivity both in healthy subjects and in diabetic patients. Plasma ***adiponectin*** levels have been reported to be decreased in some insulin-resistant states, such as ***obesity*** and ***type*** 2***

diabetes mellitus, and also in patients with coronary artery disease. On the contrary, chronic renal failure, type 1 diabetes and anorexia nervosa are associated with increased plasma ***adiponectin*** levels. Concentrations of ***plasma*** ***adiponectin*** have been shown to correlate negatively with glucose, insulin,

triglyceride levels and body mass index, and positively with high-density ***lipoprotein*** -cholesterol levels and insulin-stimulated glucose disposal. Weight loss and therapy with thiazolidinediones increased endogenous ***adiponectin*** production in humans. ***Adiponectin*** increases insulin sensitivity by increasing tissue fat oxidation, resulting in reduced circulating fatty acid levels and reduced intracellular triglyceride contents in liver and muscle. This protein also suppresses the expression of adhesion molecules in vascular endothelial cells and cytokine production from macrophages, thus inhibiting the inflammatory processes that occur during the early phases of atherosclerosis. In view of these data, it is possible that hypoadiponectinemia may play a role in the development of atherosclerotic vascular disease. In summary, the ability of ***adiponectin*** to increase insulin sensitivity in conjunction with its anti-inflammatory and anti-atherogenic properties have made this novel adipocytokine a promising therapeutic tool for the future, with potential applications in states associated with low plasma ***adiponectin*** levels.

L28 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2004:296189 CAPLUS

DOCUMENT NUMBER: 141:393195

TITLE: Study of correlation between serum adiponectin level and insulin sensitivity in patients with insulin resistance syndrome

AUTHOR(S): Hong, Jie; Gu, Weiqiong; Zhang, Yifei; Tang, Jinfeng; Yang, Ying; Chen, Yuhong; Sun, Shouyue; Zhao, Yongjie; Ning, Guang

CORPORATE SOURCE: Department of Endocrinology and Metabolism, Shanghai Institute of Endocrine and Metabolic Disease, Ruijin Hospital, Shanghai Second Medical University, Shanghai, 200025, Peop. Rep. China

SOURCE: Zhonghua Neifenmi Daixie Zazhi (2003), 19(3), 173-176

CODEN: ZNDZEK; ISSN: 1000-6699

PUBLISHER: Shanghai Shi Neifenmi Yanjiuso

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The correlation between ***adiponectin*** and insulin sensitivity, blood glucose, insulin, lipids etc was studied. Insulin sensitivity index (SI) was assessed by the reduced sample no. of Bergman's minimal model method with i.v. glucose tolerance test in subjects of normal controls, ***obesity***, impaired glucose tolerance (IGT) and ***type*** 2***

diabetes mellitus (DM). Meanwhile the anthropometrical parameters such as body mass index (BMI) and waist hip ratio (WHR) were evaluated and the serum lipids, fasting and postprandial 2 h plasma glucose and insulin levels were also measured. Compared with normal control group, the SI in obesity, IGT and type 2 DM groups was significantly decreased ($P < 0.001$), and there was no difference between those abnormal groups. Serum ***adiponectin*** level in normal control group was significantly higher than those of obese, IGT and type 2 DM groups ($P < 0.001$), and ***adiponectin*** level in type 2 DM was significantly lower than those of obese and IGT groups ($P < 0.01$, $P < 0.05$), but there was no significant difference between obese group and IGT

group. Serum ***adiponectin*** level was pos. correlated with SI and neg. correlated with waist circumference, BMI, WHR, fasting ***plasma*** glucose, postprandial 2 h ***plasma*** glucose and serum ***triglyceride*** level. In a general multivariate regression, the SI, fasting blood glucose, and WHR were significantly independent determinants for serum ***adiponectin*** concn. (all $P < 0.05$). In patients with insulin resistance syndrome, the serum ***adiponectin*** level is significantly decreased, esp. in type 2 DM patients, and ***adiponectin*** is pos. correlated to SI but neg. correlated with WHR and FBG.

L28 ANSWER 8 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003089836 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12601307
TITLE: Adiponectin and resistin--new hormones of white adipose tissue.
COMMENT: Comment in: Med Sci Monit. 2003 Nov;9(11):LE26. PubMed ID: 14586285
AUTHOR: Beltowski Jerzy
CORPORATE SOURCE: Department of Pathophysiology, Lublin Medical University, Lublin, Poland.. patfiz@asklepois.am.lublin.pl
SOURCE: Medical science monitor : international medical journal of experimental and clinical research, (2003 Feb) 9 (2) RA55-61. Ref: 51
JOURNAL CODE: 9609063. ISSN: 1234-1010.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030226
Last Updated on STN: 20030419
Entered Medline: 20030418
AB ***Adiponectin*** and resistin are recently described secretory products of adipose tissue. ***Adiponectin*** is secreted by fat cells and circulates in the blood. Plasma ***adiponectin*** concentration is reduced in obese animals and humans and in patients with type 2 diabetes mellitus. ***Adiponectin*** stimulates fatty acids oxidation, decreases ***plasma*** ***triglycerides***, and improves glucose metabolism by increasing insulin sensitivity. In addition, ***adiponectin*** inhibits the inflammatory process and possibly atherogenesis by suppressing the migration of monocytes/macrophages and their transformation into foam cells. Plasma ***adiponectin*** is lower in patients with ischemic heart disease than in body mass index-matched healthy individuals. Hypoadiponectinemia may contribute to insulin resistance and accelerated atherogenesis associated with obesity. Resistin/FIZZ3 is a member of the newly discovered cysteine-rich secretory protein family, referred to as 'resistin-like molecules' (RELM) or 'found in inflammatory zone' (FIZZ), together with FIZZ1/RELMalpha and FIZZ2/RELMbeta. Each of these has unique tissue distribution. Both resistin and FIZZ1/RELMalpha are expressed in adipose tissue. Initial studies in rodents suggested that resistin is upregulated in obesity and may be involved in the development of insulin resistance. Later studies failed to confirm this hypothesis and demonstrated reduced resistin expression in adipose tissue of obese animals. In human adipose tissue resistin is detectable at a very low level, and there is no relationship between resistin expression and obesity. Although the role of resistin in linking human ***obesity*** with ***type*** ***2*** ***diabetes*** is thus questionable, this protein is detected in peripheral blood monocytes,

L28 ANSWER 9 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:387248 BIOSIS
DOCUMENT NUMBER: PREV200200387248
TITLE: Resistin and adiponectin expression in lean and obese Zucker rats.
AUTHOR(S): Blaylock, Matthew L. [Reprint author]; Nagy, Tim R.
[Reprint author]
CORPORATE SOURCE: Nutrition Sciences, University of Alabama at Birmingham, 1675 University Blvd, Birmingham, AL, 35294, USA
SOURCE: FASEB Journal, (March 20, 2002) Vol. 16, No. 4, pp. A603. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology. New Orleans, Louisiana,

USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.

DOCUMENT TYPE: Conference; (Meeting)

LANGUAGE: English

ENTRY DATE: Entered STN: 17 Jul 2002

Last Updated on STN: 17 Jul 2002

AB The mechanisms underlying ***obesity*** and ***type*** ***2*** ***diabetes*** remain to be elucidated. Two novel adipose-derived cytokines, resistin and ***adiponectin***, have been implicated in these processes. The purpose of this study was to determine the expression of resistin and ***adiponectin*** in lean and fatty Zucker rats over a range of ages. Animals (n=9-10/group) were euthanized at 6, 7, 10, and 14 weeks of age and epididymal white adipose tissue was collected. The results showed that the fatty rats weighed significantly more, had greater adipose tissue mass as well as higher levels of ***plasma*** leptin, insulin, free-fatty acids, and ***triglycerides*** (p<0.05). Within each age class, the expression of resistin and ***adiponectin*** was reduced in the fatty compared to the lean Zucker rats (p<0.05). Our results are in agreement with recently published data suggesting that the expression of resistin and ***adiponectin*** is reduced with obesity and increasing insulin resistance.

=> d his

(FILE 'HOME' ENTERED AT 15:57:21 ON 18 DEC 2004)

FILE 'MEDLINE, BIOSIS, CAPLUS, SCISEARCH' ENTERED AT 15:57:37 ON 18 DEC 2004

L1 10959 S (C1Q OR ADIPOQ OR APM1 OR (ACRP(W)30) OR CEREBELLIN OR MULTIM
L2 83 S LIPOLYSIS(W)STIMULATED(W)RECEPTOR
L3 6417 S OBESITY(S)(TYPE(W)(2 OR II)(W)DIABETES)
L4 1992 S L3(P)(TREATMENT OR THERAPY OR THERAPEUTIC OR TREATING OR ADM
L5 6 S L4 AND L1
L6 2 DUP REM L5 (4 DUPLICATES REMOVED)
L7 3392 S ADIPONECTIN
L8 58 S L7 AND L4
L9 7407 S DIETARY(W)LIPID?
L10 33 S L9(S)PARTITION?
L11 0 S L10 AND L8
L12 0 S L8 AND L9
L13 0 S L8 AND L2
L14 74531 S PLASMA(S)(LIPOPROTEIN OR TRIGLYCERIDE?)
L15 5 S L8 AND L14
L16 2 DUP REM L15 (3 DUPLICATES REMOVED)
L17 37798 S OBESITY(S)(DISORDER OR DISEASE)
L18 237 S (ADMINISTER? OR DRUG OR DOSE OR THERAPEUTIC)(S)L7
L19 107 S L18 AND DIABETES
L20 72 DUP REM L19 (35 DUPLICATES REMOVED)
L21 22 S ADMINISTER?(S)L7
L22 5 S L20 AND L21
L23 5 DUP REM L22 (0 DUPLICATES REMOVED)
L24 0 S L20 AND PY<=1998
L25 0 S L18 AND PY<=1998
L26 213 S L7(P)L14
L27 20 S L26 AND L3
L28 9 DUP REM L27 (11 DUPLICATES REMOVED)

=>